were normal, and thus an exploratory laparotomy was not believed to be justified. Diazoxide therapy controlled her hypoglycemic symptoms by suppressing insulin secretion, although she developed hirsutism and edema due to diazoxide (3).

After diazoxide treatments were withdrawn for 2 days, the patient was hospitalized for a C-peptide suppression test. Her plasma glucose concentration before the study was 46 mg/dL, too low to do the standardized C-peptide suppression test (4, 5). Therefore, her plasma glucose concentration was "clamped" at 100 mg/dL with the aid of a Texas Instruments #59 handheld computer and PC 100 C printer (Texas Instruments, Inc., Dallas, Texas) and the PACBERG glucose clamp computer program (1) during administration of regular insulin at two dosages, 0.1 and 0.2 U/kg body weight · h. Glucose clamping was necessary to prevent severe hypoglycemia. During euglycemia, hyperinsulinemia failed to suppress endogenous C-peptide secretion, showing autonomous insulin secretion (Figure 1). These data were believed to warrant invasive procedures for insulinoma localization, and transhepatic catheterization and serial sampling of the pancreatic venous effluent were done. A doubling of the plasma insulin concentration was found at the confluence of the superior mesenteric and splenic veins, suggesting a lesion in the head of the pancreas. At laparotomy, a 1.5-cm insulinoma was resected from the uncinate process of the pancreas. The patient was cured of hypoglycemic attacks and has since returned to full employment.

Autonomous insulin release is characteristic of an insulinoma and is usually shown by the standard C-peptide suppression test (4, 5). Because C-peptide is secreted in equimolar quantities with insulin, its measurement in the plasma reflects endogenous insulin release (6). The normal physiologic response to increasing plasma insulin concentration is a decrease in endogenous insulin secretion and, therefore, C-peptide secretion (7, 8). In contrast, an insulinoma releases insulin and C-peptide autonomously, even during infusion of high concentrations of exogenous insulin (2, 4). As shown by our patient, a major problem with the exogenous infusion of insulin is the exacerbation of hypoglycemia.

Inhibition of endogenous insulin secretion by exogenous insulin infusion in normal humans has been shown by the C-peptide assay in conjunction with the euglycemic clamp technique (7, 8). This technique involves maintenance of the plasma glucose concentration at a predetermined value by simultaneously infusing both glucose and insulin intravenously. We reasoned that this method would be safe for the investigation of insulin secretion in our patient because we could avoid the risk of hypoglycemia while still assessing the effect of exogenous insulin on C-peptide secretion and, by inference, insulin. This approach was facilitated by the recent availability of a computer program to aid in achieving a clamped plasma glucose concentration (1). Before this patient, we had had no experience in performing a glucose clamp, which can be complex (9). However, with the PACBERG program, we readily performed this technique, as shown in Figure 1. Alternatively, we reasoned that a bolus dose of intravenous glucose could not be used, because the patient would secrete endogenous insulin in response to the hyperglycemia and the resulting plasma insulin and C-peptide concentration would be uninterpretable. Thus, when the biochemical data are conflicting and hypoglycemia frequent, the computerized glucose clamp provides a safe and easy approach to establishing the diagnosis before laparotomy.

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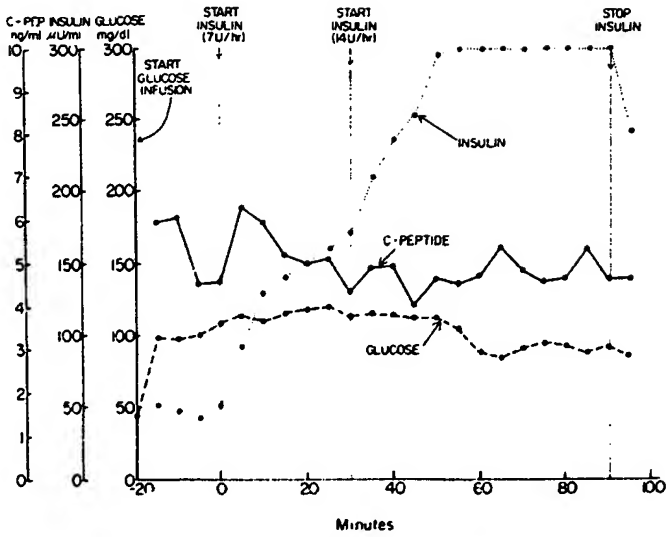

Figure 1. Graph of computerized euglycemic glucose clamp and C-peptide (*C-PEP*) suppression test in a patient with insulinoma. The high plasma insulin concentration during exogenous insulin infusion failed to suppress C-peptide release, thereby showing autonomous secretion of endogenous insulin. The plasma glucose concentration was maintained at approximately 100 mg/dL by varying the rates of glucose infusion as determined by the minicomputer.

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Spontaneous Rupture of the Stomach in a Healthy Adult Man After Sodium Bicarbonate Ingestion

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SPONTANEOUS RUPTURE of the stomach is a rare event that usually is fatal. We report the case of a patient in

whom rupture occurred within 1 minute after ingestion of a label-recommended dose of sodium bicarbonate. The patient had surgery and survived. This is the fifth such case reported in the literature.

A 31-year-old white man was apparently in excellent health before the present illness. He had no history of peptic ulcer disease, abdominal trauma, or surgery. At approximately 2000 h on 25 June 1981, the patient began eating dinner at a Mexican restaurant. He had two margueritas, an order of nachos, and a large combination plate. Because of the size of the meal, he did not have dessert but did drink a cup of coffee. After arriving home at about 2200 h, the patient noted an uncomfortable feeling of fullness in the abdomen. Because he could not find any Alka-Seltzer (Miles Laboratories, Elkardt, Indiana), he went to the refrigerator and removed a box of baking soda, a product he remembered could be used for indigestion (although he had not taken it before). He placed ½ teaspoon of baking soda in ½ glass of water and quickly drank the solution.

Within 1 minute after drinking the bicarbonate, the patient had severe abdominal pain without nausea or retching. Believing that he could induce vomiting to relieve the pain, he went to the bathroom to attempt to vomit. By this time, however, the pain was so intense that he could no longer stand erect, so he lay down on the floor. He then noted that his abdomen was distended, and he released his belt from the buckle. He called for help from his fiancee, who immediately recognized his dire condition and called the rescue squad. The patient was rushed to the hospital. At no time before entering the hospital did the patient eructate, retch, or vomit.

At a local hospital, the patient was believed to have either a Boerhaave rupture of the esophagus or perforated peptic ulcer and was taken to the operating room for exploratory laparotomy. At operation, the peritoneal cavity was noted to contain a large amount of coffee-ground material containing beans and other food particles. After vigorous irrigation, a 5-cm linear perforation was noted along the lesser curvature of the stomach, beginning near the esophagogastric junction. The stomach was noted to contain a "massive" amount of retained food. No evidence of ulceration or other abnormality was seen, and the perforation was oversewn in two layers. Histologic examination of the margins of the involved stomach wall revealed "full thickness hemorrhage and focal necrosis, but relatively little attendant acute inflammation."

The patient tolerated the procedure well, but his postoperative course was complicated by the development of a right subphrenic abscess, subhepatic abscess, and respiratory failure believed secondary to adult respiratory distress syndrome. After 21 days in the hospital, he was discharged and has continued to do well.

"Spontaneous" rupture of the stomach is an uncommon condition—only 71 cases have been reported in the English literature. Six of these were associated with antacid ingestion (1-6); only four reports specified sodium bicarbonate as the antacid (1-4).

Presumably, the rupture is caused by increasing intragastric pressure secondary to distention of the stomach by any combination of solid, liquid, or gas. In addition to the previous cases of gastric rupture associated with sodium bicarbonate ingestion, several cases in the literature suggest that fermentation might have occurred in the stomach (7, 8), producing large quantities of gas leading to gastric distention and rupture. This pathogenesis is similar to that in the present case—ingestion of sodium bicarbonate resulted in the rapid production of enough carbon dioxide gas to rupture a stomach already distended with food.

Spontaneous rupture of the stomach is associated with a high mortality rate. In Albo and colleagues' series (9), there was an 85% mortality rate; however, 26 of 44 patients did not undergo surgery, and all of the them died. Of the 18 patients who did have surgery, there was a 65% mortality rate. In the 30 cases reported since Albo and colleagues' series, all patients had surgery, and 11 survived, producing a 63% mortality rate.

We present the fifth case of spontaneous rupture of the stomach associated with sodium bicarbonate ingestion. Our patient was uncomfortable until he had ingested sodium bicarbonate, at which time he became acutely ill. We believe that his initial discomfort was due to "overdistention" of the stomach and that his acutely ill state was due to gastric rupture secondary to release of carbon dioxide by the reaction of sodium bicarbonate with gastric acid. (The kinetics of this reaction will be discussed in a later report.) Because of the high mortality rate associated with this lesion and because of the availability of effective antacids without gas production as a side effect, we believe that the use of sodium bicarbonate should be discontinued for the treatment of heartburn or other gastrointestinal symptoms.

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Possible Interaction Between Amiodarone and Phenytoin

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AMIODARONE, an investigational antiarrhythmic drug, has been reported to be effective in treating various cardiac tachyarrhythmias (1). Pharmacokinetic interactions with digoxin, warfarin, and class I antiarrhythmic drugs have been reported during treatment with amiodarone